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=> s protein tyrosine phosphoryla? (3a) 36
L1 4 PROTEIN TYROSINE PHOSPHORYLA? (3A) 36

=> s l1 or PTP36 or PTP 36
L2 38 L1 OR PTP36 OR PTP 36

=> s l2 and (transgen? or knockout or disrup? or deficie? or delet?)
L3 14 L2 AND (TRANSGEN? OR KNOCKOUT OR DISRUP? OR DEFICIE? OR DELET?)

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L4 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2003 ACS
AN 2002:794361 CAPLUS
DN 137:305753
TI ***Transgenic*** mice containing ***PTP36*** tyrosine phosphatase gene ***disruptions*** and uses in screening drug
IN Allen, Keith D.
PA USA
SO U.S. Pat. Appl. Publ., 30 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2002152493	A1	20021017	US 2001-5467	20011204
WO 2002045500	A2	20020613	WO 2001-US47566	20011205

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, US, UZ, VN, YU, ZA, ZM, ZW
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PRAI US 2000-251796P P 20001206
AB The present invention provides ***transgenic*** mice comprising a ***disruption*** in a ***PTP36*** tyrosine phosphatase gene and methods for the characterization of ***PTP36*** tyrosine phosphatase gene function. Specifically, the present invention provides ***transgenic*** mice comprising mutations in a ***PTP36*** gene. Such ***transgenic*** mice are useful as models for disease and for identifying agents that modulate gene expression and gene function, and as potential treatments for various disease states and disease conditions.

L4 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 2002:369580 BIOSIS
DN PREV200200369580
TI Neuroplastic behavior at the ganglion level: Comparative studies between (mRen2)27 and mRen2.Lewis models of hypertension.
AU Logan, Exazevia Montreal (1); Diz, Debra I.; Averill, David B.; Ferrario, Carlos M.; Ganten, Detlev; Aileru, Azeez A. (1)
CS (1) Life Sciences, Winston Salem State University, 115 South Chestnut St., Winston-Salem, NC, 27101 USA
SO FASEB Journal, (March 22, 2002) Vol. 16, No. 5, pp. A875. http://www.fasebj.org/. print.
Meeting Info.: Annual Meeting of Professional Research Scientists on Experimental Biology New Orleans, Louisiana, USA April 20-24, 2002
ISSN: 0892-6638.
DT Conference
LA English
AB Synaptic plasticity in superior cervical ganglia isolated from hypertensive (mRen2)27 ***transgenic*** rats is increased relative to Hannover Sprague-Dawley rats, as measured by decay time constants of post-tetanic potentiation (PTP) and long-term potentiation (LTP) after a tetanizing volley (20hz/20s) in the presence of hexamethonium. A new line of congenic hypertensive rats, developed by transferring the mouse renin gene to the inbred Lewis background (mRen2.Lewis), is hypertensive and has many but not all the same characteristics of the parent strain. Angiotensin II (16nM) increases the internal carotid nerve compound action potential by 14% in (mRen2)27 and by 8% in mRen2.Lewis animals. After a tetanic pulse, PTP and LTP were significantly longer (***PTP*** : ***36*** min, LTP: 1.03X103 min; n=8) in (mRen2)27 than mRen2.Lewis

(PTP: 15 min, LTP: 255 min; n=7) rats. The mechanisms responsible for these disparities in transmission are not known, but the data suggest that we can dissociate ganglionic changes occurring during sustained high blood pressure from those related to genetic differences in synaptic plasticity using these two strains of animals.

L4 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
AN 2000:228674 BIOSIS
DN PREV200000228674
TI Regulation of mouse podocyte process dynamics by protein tyrosine phosphatases: Rapid Communication.
AU Reiser, Jochen (1); Pixley, Fiona J.; Hug, Andreas; Kriz, Wilhelm; Smoyer, William E.; Stanley, E. Richard; Mundel, Peter
CS (1) Department of Medicine and Department of Anatomy and Structural Biology, Albert Einstein College of Medicine, Bronx, NY USA
SO Kidney International, (May, 2000) Vol. 57, No. 5, pp. 2035-2042.
ISSN: 0085-2538.
DT Article
LA English
SL English
AB Background: Effacement of podocyte foot processes occurs early in many glomerular diseases associated with proteinuria and is accompanied by a reorganization of the actin cytoskeleton. The molecular mechanisms regulating these structural changes are poorly understood. Methods: To address these questions, we analyzed the effect of the polycation, protamine sulfate (PS), and puromycin aminonucleoside (PA) on the morphology, cytoskeleton, and tyrosine phosphorylation of differentiated process-bearing cultured podocytes. Results: PS and PA induced similar profound morphological alterations, including retraction and detachment of podocyte processes from the extracellular matrix (ECM). The effects of PS occurred within six hours, whereas PA showed its most severe effects after 72 hours. Structural changes included reorganization of the actin cytoskeleton and focal contacts and were accompanied by an increase in tyrosine phosphorylation. The same effects were induced by application of vanadate, an inhibitor of protein tyrosine phosphatases (PTPs), suggesting that PTPs regulate podocyte process structure. Since ***disruption*** of the actin cytoskeleton with cytochalasin B protected the cells from PS-induced effacement and detachment, cytoplasmic PTPs were implicated in these events. Using reverse transcription-polymerase chain reaction (RT-PCR), we demonstrated the expression of four cytoplasmic PTPs in podocytes: SHP-2, PTP-PEST, PTP-1B, and ***PTP*** - ***36*** . Conclusions: These studies indicate an important role for cytoplasmic PTPs as regulators of podocyte process dynamics. Future studies will aim at restoring the normal foot process architecture of podocytes in glomerular diseases associated with proteinuria by modulating the activity of cytoplasmic PTPs.

L4 ANSWER 4 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2
AN 2000:296971 BIOSIS
DN PREV200000296971
TI Cytoskeletal protein tyrosine phosphatase PTPH1 reduces T cell antigen receptor signaling.
AU Han, Shulin; Williams, Scott; Mustelin, Tomas (1)
CS (1) Laboratory of Signal Transduction, La Jolla Cancer Research Center, Burnham Institute, 10901 North Torrey Pines Road, La Jolla, CA, 92037 USA
SO European Journal of Immunology, (May, 2000) Vol. 30, No. 5, pp. 1318-1325. print.
ISSN: 0014-2980.
DT Article
LA English
SL English
AB The subgroup of protein tyrosine phosphatases that contain an N-terminal ezrin-, radixin- and moesin homology (ERM) domain and a C-terminal catalytic domain is represented by three enzymes in Jurkat T cells, PTPH1, PTP-MEG1 and ***PTP36*** . These enzymes are located at the cytoplasmic face of the plasma membrane and may be involved in regulation of the membrane cytoskeleton, signal transduction, or both. Here we report that expression of PTPH1 in Jurkat T cells reduced the TCR-induced activation of reporter genes encompassing parts of the IL-2 gene promoter and driven by nuclear factor of activated T cells plus activator protein-1. PTP-MEG1 had a weaker inhibitory effect, while ***PTP36*** had none. The catalytically inactive mutants PTPH1-CS and PTP-MEG1-CS lacked effects on gene transcription. Expression of active PTPH1 also reduced receptor-induced activation of Erk2 MAP kinase, its upstream activator, Mek, and the Jnk kinases. The effect of PTPH1 was reduced by ***deletion*** of its N-terminal ERM domain. We suggest that PTPH1 inhibits T cell activation by dephosphorylating membrane-associated targets involved in TCR signaling.

L4 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 3
AN 1999:467437 BIOSIS
DN PREV199900467437
TI Regulation of phosphorylation level and distribution of ***PTP36*** , a putative protein tyrosine phosphatase, by cell-substrate adhesion.
AU Ogata, Masato (1); Takada, Tsuyoshi; Mori, Yoshiko; Uchida, Yohzo; Miki, Tsuneharu; Okuyama, Akihiko; Kosugi, Atsushi; Sawada, Motoyuki; Oh-hora, Masatsugu; Hamaoka, Toshiyuki
CS (1) Biomedical Research Center, Osaka University Medical School C6, 2-2 Yamadaoka, Suita, Osaka, 565-0871 Japan
SO Journal of Biological Chemistry, (July 16, 1999) Vol. 274, No. 29, pp. 20717-20724.
ISSN: 0021-9258.
DT Article
LA English
SL English
AB Recently we have cloned a putative protein tyrosine phosphatase, ***PTP36*** /PTPD2/pez, which possesses a domain homologous to the N-terminal half of band 4.1 protein. In mouse fibroblasts adhered to substrates, ***PTP36*** was phosphorylated on serine residues. ***PTP36*** was found to make complexes with serine/threonine kinase(s), which phosphorylated ***PTP36*** in vitro. ***PTP36*** was dephosphorylated rapidly when the cell-substrate adhesion was ***disrupted*** and it was phosphorylated again along with the reattachment of the cells to fibronectin. Rephosphorylation of ***PTP36*** seemed to depend on actin polymerization since it was inhibited by cytochalasin D. The cell detachment also induced the translocation of ***PTP36*** into the membrane-associated cytoskeletal fraction. Staurosporine and ML-9, which inhibited the phosphorylation of ***PTP36*** in vivo, induced the translocation of ***PTP36*** too. On the contrary, when the dephosphorylation of ***PTP36*** was inhibited by okadaic acid, no translocation of ***PTP36*** was induced by the celldetachment. These results demonstrate that the cell-substrate adhesion and cell spreading regulates the intracellular localization of ***PTP36*** most likely through its phosphorylation and therefore, ***PTP36*** may play important roles in the signal transduction pathway of cell-adhesion.

L4 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 4
AN 2000:88313 BIOSIS
DN PREV200000088313
TI Characterization of newly identified four isoforms for a putative cytosolic protein tyrosine phosphatase ***PTP36*** .
AU Aoyama, Koji; Matsuda, Tsukasa; Aoki, Naohito (1)
CS (1) Department of Applied Molecular Biosciences, Graduate School of Bioagricultural Sciences, Nagoya University, Furo-cho, Chikusa-ku, Nagoya, 464-8601 Japan
SO Biochemical and Biophysical Research Communications, (Dec. 20, 1999) Vol. 266, No. 2, pp. 523-531.
ISSN: 0006-291X.
DT Article
LA English
SL English
AB In the course of determining the expression profiles of protein tyrosine phosphatases in lactating mammary gland, we found the expression of an isoform for a putative cytosolic and cytoskeleton-associated protein tyrosine phosphatase ***PTP36*** . Further detailed RT-PCR and Northern blot analyses revealed the expression of several isoforms for ***PTP36*** in a tissue-dependent manner. We have cloned the cDNAs encoding four truncated isoforms for ***PTP36*** and designated ***PTP36*** -A, -B, -C, and -D, respectively. ***PTP36*** -A and -C had new sequences generated due to frameshift, whereas ***PTP36*** -B and -D were in-frame variants. Gly- and Glu-rich domains and a putative PTP domain were missing from ***PTP36*** -A, but the band 4.1 domain remained. ***PTP36*** -B retained the band 4.1 and PTP domains but lacked Pro-, Gly- and Glu-rich domains. Most domain structures were lacking in ***PTP36*** -C and -D. Interestingly, ***PTP36*** -C contained an incomplete band 4.1 domain, but the newly created sequence exhibited high homology to human nebulette, which was also suggested to associate with cytoskeletons. When transiently expressed in COS7 and HEK293 cells, not only the wild type but also all the isoforms were recovered in Triton X-100-insoluble cytoskeleton-associated fractions and this distribution was not affected by mechanical cell detachment and treatment with a kinase inhibitor staurosporine. Such cellular distribution of ***PTP36*** was also observed in stable COS7 clones. Further studies using ***deletion*** mutants suggested that the first 30 amino acids as well as the band 4.1 domain of ***PTP36*** were involved in association with Triton X-100 insoluble cytoskeletons. Tissue-dependent expression and ***deletion*** in domain structures might reflect the biological significance of the isoforms for ***PTP36*** in certain physiological conditions.

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